BLACK RABBITS ON LUNDY: TUDOR TREASURES OR POST-WAR PHONIES?

by

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ABSTRACT

Lundy is renowned for its feral black rabbits which, according to popular tradition, have inhabited the island since medieval times. Black rabbit fur was valued for much of the Middle Ages, explaining why warreners of Lundy might have favoured them, but genes responsible for feral rabbit melanism remain unexplored. Further potential complicating factors occur in the form of recent (twentieth century) small-scale domestic rabbit introductions to the Lundy feral population. To gain insight into genetic mechanisms underlying melanism on Lundy, rabbit samples were collected and subjected to molecular analysis. The Lundy rabbit population is shown to harbour non-functional copies of the agouti signalling protein (*Asip*) gene, a main determinant of coat colour in mammals. The observed genetic mutation is not unique to Lundy, having been reported to underlie dark coat colour phenotypes in various domestic rabbit breeds. The mutation is recessive and only phenotypically expressed in homozygous individuals. Although presence of this particular allele does not preclude recent genetic augmentation or replacement, simple population genetics show that allele persistence from a medieval introduction is not impossible.

Keywords: *Lundy, black rabbits, DNA analysis, melanism gene, medieval ancestry*

INTRODUCTION

Although there is fossil evidence of rabbits, *Oryctolagus cuniculus* (Linnaeus, 1758), in what was to become the British Isles during the Hoxnian (Yalden, 1999), for the most recent epoch until ~2.3ka before present they were confined to the Iberian Peninsula and, possibly, north-western Africa (Surridge *et al.* 1999). Phoenician and Roman traders transported rabbits across the Mediterranean and they were present in northern and central Europe by the Middle Ages, probably sometime after AD1000 (Lever, 1994). Normans were the first to successfully introduce rabbits to

Britain, often to its sandy or peat-covered offshore islands. There rabbits were effectively confined, could burrow easily and were safe from mainland predators (Williamson, 2007). Lundy was one of the first British locations in which their presence is recorded. Irving *et al.* (1997) cite Exeter City Archives to state that between 1183 and 1219 the tenant of Lundy was entitled to take 50 rabbits a year 'from certain chovls on the island'. By 1274 a report to Edward I (regarding the produce of Lundy) stated 'taking of rabbits is estimated at 2000, £5 10s.' The report also indicates the primary purpose of such a harvest: '5s. 6d. each hundred skins, because the flesh is not sold' (Ritchie, 1920). That Henry III could instruct the constable of Lundy to put the proceeds of the sheriff of Devon's sale of 2,500 Lundy rabbit skins 'towards the expense of building the new tower' (Powicke, 1949) demonstrates the value of such commerce. Moreover, Lundy rabbits were particularly valued because a large proportion provided black fur, for centuries favoured as clothing trim or lining by the wealthy and socially elevated. Henry VI possessed a night shirt lined with black rabbit fur (Mason and Parry, 2010) and Henry VIII was obviously preparing to obtain his own when Hampton Court accounts listed the purchase of 'a great long auger of irne, to make and bore coney holes within the kynges beries new made for blake coneys in the warren' (Williamson, 2006).

Although black rabbit fur was a desirable product (Veale, 2003) it could be one with a legally restricted clientele; Tudor sumptuary laws, for example, dictated who was privileged to wear such 'black coney' (Cox, 2006) and in the majority of mainland warrens common grey rabbits remained most numerous, their fur used for warmth rather than for display and their meat for culinary purposes (Bailey, 1988). On the other hand, in some coastal and East Anglian warrens such as Methwold ('famous to a proverb' for its black rabbits according to Blomefield (1805) and Wretham (known as 'The Black Rabbit' warren), warreners specialised in breeding black rabbits to supply the demands of fine tailoring and, particularly, millinery (Mason and Parry, 2010).

Such specialism implies sophisticated warren management and this indeed occurred. A comprehensive description of methods and equipment employed (Sheail, 1971 and Mason and Parry, 2010 for such) is beyond the province of this paper. Suffice to say, elsewhere in the British Isles warrened rabbits were usually harvested by net or non-lethal trap with assistance from (muzzled) ferret and/or dog so a conscientious warrener could dictate the sex and phenotype of those rabbits killed and those released to produce subsequent generations. Ratios of both could be controlled precisely; in Hertingfordbury (Hertfordshire) in 1634 a warren tenant was bound to leave 'one hundred and twenty female Conyes and twenty male Conyes wheirof the better halfe to be black' (Hertfordshire Archives and Local Studies D/EP T264 cited by Williamson, 2007). It is not, thus, unreasonable to posit that similar methods were utilised on Lundy to manipulate its rabbit population and thus propagate a desirable black rabbit product.

Sheail (1971) and Ritchie (1920) note that by the fifteenth and sixteenth centuries black rabbits had been introduced to several offshore islands in England and Scotland respectively and these introductions founded the black rabbit populations for which such warrens were so prized. Sheail goes on to note 'Even today, there are a few black rabbits on Lundy Island', the implication being they are a legacy of such introductions. That there are 'a few black rabbits on Lundy' is not in doubt but whether this trait was

acquired via unbroken inheritance from medieval introductions is a difficult question to address, particularly when it is known that several introductions of domestic rabbits occurred in the twentieth century, bringing potential genetic novelty to the islands feral rabbit population (Linn, 1997).

Two loci play a main role in coat colour pigmentation in vertebrates – the extension locus and the agouti locus (Hoekstra, 2006; Suzuki, 2013). The extension locus encodes the melanocortin 1 receptor *(Mc1r)*, and the agouti locus encodes the agouti signalling protein *(Asip)*. *Mc1r* determines which pigment is synthesized from the melanin precursor dopaquinone. When *Mc1r* is active, brown or black eumelanin is produced, resulting in dark fur. When *Mc1r* is inhibited, yellow or red pheomelanin is produced, giving lighter coloured fur (Hoekstra, 2006). *Asip* is an antagonist of *Mc1r*. It binds to *Mc1r*, and by doing so, inhibits *Mc1r*'s function, resulting in reduced eumelanin synthesis and therefore a lighter phenotype (Suzuki, 2013).

Mutations affecting coat colour in the extension and agouti locus have been described for rabbits. For example, a six base-pair deletion in *Mc1r* is associated with *dominant* black coat colour in this species (Fontanesi *et al.*, 2006). This 6 basepair (bp) deletion seems to hamper effective binding of *Asip* to *Mc1r* and thus prevents its inhibition, resulting in a black phenotype.

A 1bp insertion in *Asip* has been associated with *recessive* black coat colour in rabbits (Fontanesi *et al.*, 2010). This deletion in the 2nd exon of the gene causes a frameshift and introduces preliminary stop codons, i.e. it is a loss-of-function mutation (Figure 1). As with the 6bp deletion in the extension locus, this mutation prevents the inhibition of *Mc1r* and therefore results in a black phenotype.

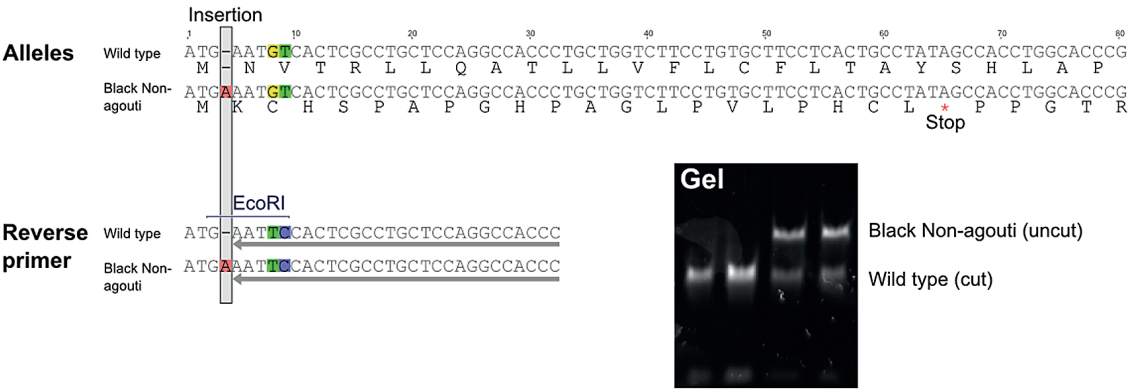
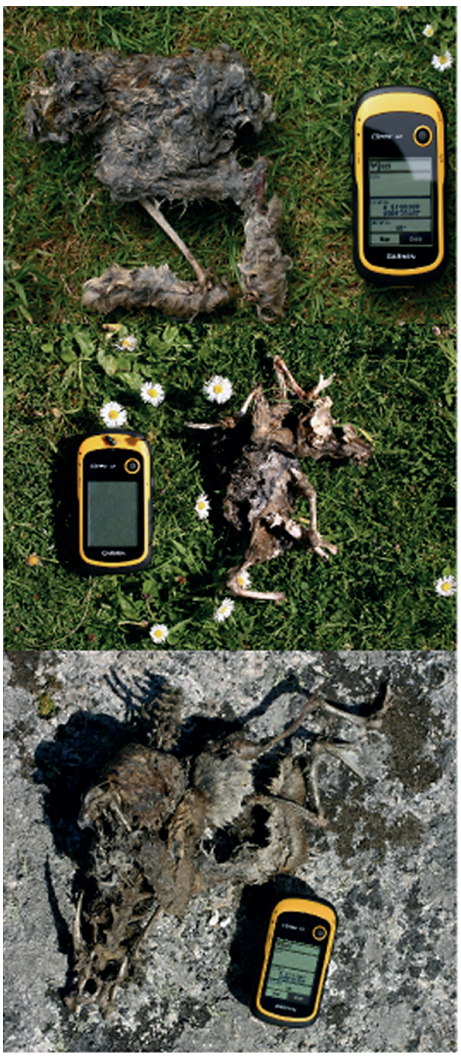


Figure 1: PCR-RFLP essay of Fontanesi *et al*. (2016) applied here.
Alleles: examples of DNA and amino acid sequences of a wild-type and a black non-agouti allele (partial). The 1bp insertion in the black non-agouti allele causes a frameshift that introduces stop codons resulting in a truncated protein.
Reverse primer: the reverse primer Ex2-ART-R (grey arrows) introduces an artificial EcoRI restriction site in the wild-type allele only.
Gel: the gel image shows the banding patterns of the four 2016 samples. Two samples on the left: homozygous wild-type; two samples on the right: heterozygous, carrying a wild-type and a black non-agouti allele

Both black coat mutations have been described for domestic breeds, but, to our knowledge, are currently not known from wild rabbit populations. The low frequency of black rabbits on Lundy might suggest recessive inheritance of the trait. With this in mind, we investigate whether the 1bp frameshift mutation in *Asip* underlies the black rabbit phenotype that is observed on the island.

MATERIAL AND METHODS

Rabbit tissue samples were obtained from dead rabbits found at various locations on Lundy in June 2016 and June 2017 in an *ad hoc* manner (Table 1, Figure 2). Soft tissue was taken from rabbit remains using forceps and dissecting scissors where possible. Where this was not possible, fur was sampled. Eight wild-type rabbits were sampled in 2016; 28 wild-type and 2 black rabbits were sampled in 2017. Carcass location coordinates were noted via GPS for most of the samples collected in 2017. Carcasses were photographed to record phenotype and tissue/fur samples were stored in absolute ethanol until further processing.



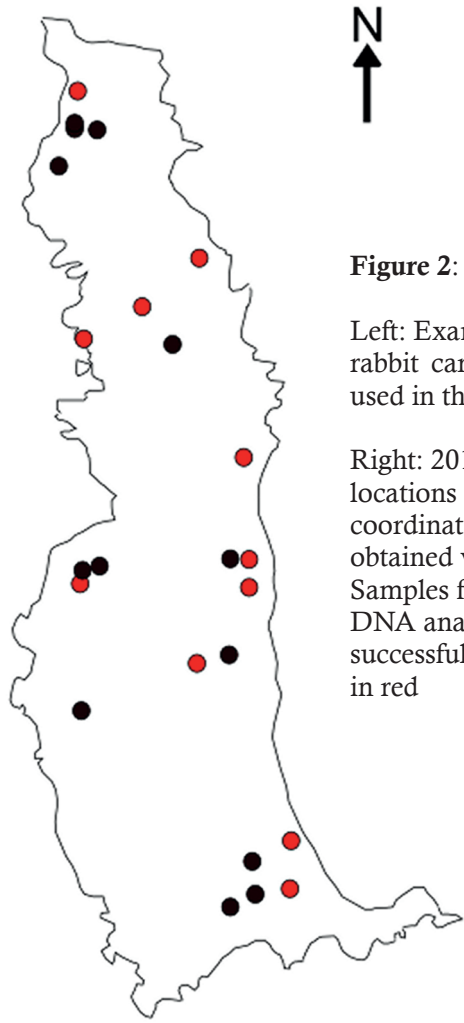


Figure 2:

Left: Examples of rabbit carcasses used in this study

Right: 2017 sample locations for which coordinates were obtained via GPS. Samples for which DNA analysis was successful are given in red

Table 1: Samples collected in June 2016 and June 2017.
Data of collection and coordinates of carcasses sampled are given when available. na=not available. Phenotype indicates phenotype of the specimen: W=wild-type, B=black. Genotype indicates the genotype of the specimen: hom. wt=homozygous wild-type, heteroz=heterozygous, hom. na=homozygous non-agouti

Date	Sample	Degree	Minutes	Degree	Minutes	Location	Phenotype	Genotype
na	T1					600m North of North Quarry	W	hom.wt
na	T2					500m North of North Quarry	W	
10/06/2016	T3					Jenny's Cove	W	
09/06/2016	T4					Lundy Village	W	hom. wt
09/06/2016	T5					Lundy Village	W	heteroz.
10/06/2016	T6					Jenny's Cove	W	
na	T7					South east path	W	heteroz.
09/06/2016	T8					900m North of North Quarry	W	
06/06/2017	1A	51	9.807	4	39.659	NW of Castle	W	hom. wt
06/06/2017	1B	51	10.642	4	40.543	E of Earthquake	W	hom. wt
06/06/2017	1C	51	11.283	4	40.166	S of Threequarter Wall	B	
06/06/2017	1D	51	11.373	4	40.290	S of Threequarter Wall	W	hom. wt
06/06/2017	1E	51	11.853	4	40.484	North End	W	
07/06/2017	2A	51	9.936	4	39.655	By Millcombe chairs	W	
07/06/2017	2B	51	9.939	4	39.647	E Coast path just N of Millcombe	W	hom. wt
07/06/2017	2C	51	10.643	4	39.833	E Coast path just N of VC Quarry	W	
07/06/2017	2D	51	10.700	4	39.841	E Coast path between VC and North Quarries	W	hom. wt
07/06/2017	2E	51	10.969	4	39.861	Low E coast path 500+m N of North Quarry	W	hom. wt
07/06/2017	2F	51	10.977	4	39.857	Low E coast path 600+m N of North Quarry	W	
07/06/2017	2G	51	11.508	4	40.050	Low E coast path 100m N of Mousehole and Trap	W	hom. wt
09/06/2017	3A	51	11.950	4	40.561	North End, Long Roost	W	hom. wt
09/06/2017	3B	51	11.290	4	40.546	North End, Long Roost	W	hom. wt
09/06/2017	3C	51	11.868	4	40.587	Just S of Long Roost	W	
09/06/2017	3D	51	11.856	4	40.583	Further S of Long Roost	W	
09/06/2017	3E	51	11.759	4	40.648	Further S of Long Roost	W	
10/06/2017	4A	51	10.672	4	40.475	100m NW of St Helena's Church	W	
11/06/2017	5C	51	10.626	4	39.838		W	
11/06/2017	5D	51	10.626	4	39.838		W	hom. wt
11/06/2017	5E	51	10.442	4	39.920		W	
11/06/2017	5F	51	9.871	4	39.825		W	
11/06/2017	5G	51	9.797	4	39.811		W	
11/06/2017	5H	51	9.760	4	39.912		W	
14/06/2017	6A	51	10.419	4	40.059		W	hom. wt
14/06/2017	6B	51	10.617	4	40.559		W	
14/06/2017	6C	51	10.288	4	40.549		W	
na	7A						W	heteroz.
na	8A						W	
21/05/2017	0P					Lundy Village	B	hom. na

DNA was extracted using the Qiagen Blood and Tissue kit. PCR was employed to amplify small fragments of the ASIP gene. PCR reactions used primers described in Fontanesi *et al.* (2010). PCR was performed on the samples collected in 2016 using primers Ex2-F and Ex2-R (Fontanesi *et al.*, 2010), targeting a fragment of 396bp (397bp if insertion is present) (including primers) that contains part of intron 1, exon 2 and part of intron 2. PCR products were sent to the Natural History Museum London for Sanger sequencing. A second PCR was performed using primers Ex2-F and Ex2-ART-R, targeting a fragment of 94bp (95bp if insertion is present) (including primers). Primer Ex2-ART-R introduces a restriction site for the endonuclease EcoRI (G^AATTC) in the wild-type allele only i.e. when the insertion in exon 2 is absent (Figure 1) (Fontanesi *et al.*, 2010). This latter PCR was performed on all samples and the products were subsequently digested with EcoRI. Reactions were performed at 37°C for one hour in a total volume of 20µl and used the following reaction mixture: 8µl PCR product, 20 units EcoRI-HF (NEB), 40µg BSA. Samples were separated on 5% Mini-PROTEAN® TBE Precast Gels (BioRAD). GelRed was used for staining. Samples were run at 100 volts for 45 minutes and visualised under UV light. Genotypes were scored by hand.

Genotypes frequencies were tested for Hardy-Weinberg equilibrium using a chi-square test available on the OEGE website (www.oege.org/software/hwe-mr-calc.shtml) (Rodriguez *et al.*, 2009).

RESULTS

PCR product was obtained for 3 of the 8 soft tissue samples collected in 2016 using the Ex2-F and Ex2-R primers (the 396/397bp fragment). The products were sent for sequencing and for one sample readable Sanger trace files were obtained (sample T5: wild-type phenotype). The other 2 sequencing reactions failed (i.e. unreadable Sanger trace files were obtained), most likely due to the PCR products being of poor quality. Visual inspection of the T5 trace files revealed the specimen to be heterozygous and to carry a wild-type and a recessive black non-agouti allele.

All 38 samples (Table 1) were subsequently subjected to PCR-RFLP analysis (the 94/95bp fragment). PCR product was obtained for 4 of the samples collected in 2016 and 13 samples collected in 2017, which were digested with EcoRI. Two of the 2016 specimen were homozygous for the wild-type allele and two heterozygous (i.e. carried the recessive non-agouti allele in addition to the wild-type allele) (Figure 1). This observation does not deviate from Hardy-Weinberg expectations (Chi-square test: χ^2=0.44, p>0.05). Using the observed allele frequencies 2 homozygous wild-type, 1.5 heterozygous and 0.25 homozygous black rabbits would have been expected for a population that is in Hardy-Weinberg equilibrium. Eleven of the 2017 specimen were homozygous for the wild-type allele, one was heterozygous, and one homozygous for the recessive non-agouti allele. As expected, this latter homozygous specimen had the black phenotype. It was the only black individual for which genotype data were obtained. However, the chi-square test rejected the Ho hypothesis of this sample being in Hardy-Weinberg equilibrium (χ^2=5.05, p<0.05). Here, 10.2 homozygous wild-type rabbits, 2.65 heterozygous rabbits and 0.17 homozygous black rabbits would have been expected for a population that is in Hardy-Weinberg equilibrium.

DISCUSSION

This study shows that at least some black rabbits on Lundy derive their black coat colour from a 1bp insertion in the *Asip* gene. This insertion is a frameshift mutation that makes the gene product non-functional (Fontanesi *et al.*, 2010). As a consequence *Asip* will not inhibit *Mc1r* and the relative amount of black eumelanin increases. This mutation is a recessive mutation; the black phenotype is only expressed in individuals that carry two loss-of-function alleles. It is, to our knowledge, the first time that this specific mutation has been reported within a wild rabbit population.

Rabbits have been present on Lundy since medieval times during which black specimens were highly valued. A question arises whether the black rabbits of today are direct descendants of the black rabbits that were bred when Lundy acted as a medieval warren, and how they are related to black specimens in other populations and breeds. The 1bp insertion observed here is identical to the one reported for domestic breeds (Fontanesi *et al.*, 2010) and it seems highly unlikely that the Lundy non-agouti allele arose independently from the one observed in those breeds. It is more likely that Lundy and domestic rabbits obtained the allele from a single common ancestor. The finding of this specific allele on Lundy therefore supports a medieval origin of the black phenotype of present day domestic breeds.

It is also possible, however, that the black non-agouti allele arrived on Lundy in much more recent times. Albeit insular, the Lundy population cannot be considered closed. It has been restocked with specimens from other localities on numerous occasions in the twentieth century. These restocking exercises also involved domestic animals, including for example dark-coloured specimens of the Rex breed (Linn, 1997). Interestingly, recent genetic research has shown that dark coat colour in this specific breed is determined by the same frameshift mutation in *Asip* (Yang *et al.*, 2015). It is therefore not impossible that the black non-agouti allele observed here originated from Rex or other animals introduced on the island only a few decades ago. In that case, another more ancient 'black allele' might still linger within the Lundy genepool. More research, that also for example includes the *Mc1r* locus, is needed to answer this.

On the UK mainland organised warrening eventually fell prey to a variety of changes in agriculture, practices associated with hunting and legislation. Agricultural changes extended the range of habitat available for escaped rabbits to set up their own, feral populations. New game laws enabled farmers to exploit rabbits on their own land whilst selective elimination of 'ground game' predators favoured rabbit proliferation. Localities varied in their susceptibility and timescale associated with such changes (Williamson, 2007) but by the nineteenth century feral rabbit numbers had increased to the extent they exercised significant ecological influence over much of the mainland and were considered a serious agricultural pest by some. In 1840 a Select Committee was set up to look into the matter (Sheail, 1971). Rabbit proliferation continued despite attempts at control and by the 1950s it was estimated (Thompson and Worden, 1956) that the British mainland population was 60-100 million, with densities of up to 35-50ha^{-1}. As warrens declined, their rabbits, where not deliberately eliminated, spread and mingled with feral relatives and previously-selected phenotypic characters were lost via introgression and natural selection. Presumably, the Lundy warren followed a similar fall from grace. Preponderance of black rabbits whilst Lundy was managed as a warren is unlikely to have resulted from a naturally-selected Hardy-Weinberg

equilibrium. Evidence from UK mainland warrens indicates intensive management was required to maintain the black phenotype. As warrening decreased and feral populations increased, alleles responsible for black phenotype will have dispersed into the feral genepool to produce a new Hardy-Weinberg equilibrium. It is likely that other anthropogenic influences such as introduction of non-native zoonoses (myxomatosis and haemorrhagic virus), and culling will have produced low population numbers prone to genetic bottlenecking, also with consequences for subsequent Hardy-Weinberg outcomes.

It is not currently possible to establish beyond doubt whether Lundy's black rabbits' alleles result from medieval selection or from twentieth century introductions. One point is, however, worth making. The melanism allele located in this study is recessive. This has two functional consequences, one genetic and one historical. Both might have a bearing on the matter.

In 1992 myxomatosis reduced the Lundy rabbit population to a few hundred animals (Compton *et al.*, 2004), and since then the population has gone through at least three more virus induced bottlenecks (Compton *et al.*, 2007), including that of 2017, yet the melanistic trait persists. Although only a small number of Lundy rabbits express the black phenotype (in the 2017 sample 2 out of 30 rabbits were black), a much larger number will be heterozygous and carry the black non-agouti allele. Assuming the population was in Hardy-Weinberg equilibrium in 2017 (which according to the genotype analysis for currently unknown reasons might not be the case – see results), the model predicts that 38% of the rabbits will have been heterozygous and 26% of the alleles on Lundy will have been black non-agouti. This illustrates an effect of heterozygotic trait possession: although homozygotes may be rare or even absent, heterozygote frequencies are much higher and it is they that are most likely to carry the trait forward for future generations to express. So, depending on allele frequencies, a recessive trait may be rare in expression but robust in the face of genetic bottleneck.

The same characteristic might also determine which type of melanism would be selected (albeit unknowingly) by warreners. For producing pure-breeding melanistic rabbits a recessive trait might be preferred. Dominant alleles mask non-desirable wild-type alleles but recessive traits require both alleles to conform before the trait is expressed and ensures pure-breeding strains will persist.

Neither of these factors can determine gene provenance with certainty but they may aid the suggestion that for medieval melanism genes to have survived to the present day in Lundy rabbits is not entirely unfeasible. Equally, however, it does not preclude the possibility of other, more ancient, genes persisting and it is for these that the search continues …

ACKNOWLEDGEMENTS

We would like to thank Tom Dickins (Middlesex University) for organising field trips to Lundy and for useful discussions regarding melanism in animals. We thank Chris Pawson (University of the West of England) for collecting a black rabbit sample. Fieldwork was funded by Middlesex University (Faculty of Science and Technology).

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